Toxic effects in dairy cattle following the ingestion of a large volume of canola oil

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Abstract — The clinical and laboratory findings of a group of 9 dairy cattle that accidentally ingested large volumes of canola oil are described. Four of the animals died, and 3 were necropsied. No specific cause of death was found, although a number of theories are advanced. This is the first report of such an occurrence.

Résumé — Effets toxiques de l'ingestion d'une grande quantité d'huile de colza par des bovins laitiers. Les résultats de l'examen clinique et des tests de laboratoire effectués pour un groupe de 9 bovins laitiers qui ont accidentellement ingéré de fortes quantités d'huile de colza sont présentés. Quatre des animaux sont morts et trois ont fait l'objet d'une nécropsie. Aucune cause précise n'a été établie pour la mort, mais des théories ont été proposées. C'est la première fois qu'un tel événement est signalé.

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Canola (*Brassica napus*) meal is now recognized as an important component of dairy cattle rations. Canola oil can also be used as a source of energy (especially in early lactation, although it should not form more than 1% to 2% of the daily ration (200 to 400 mL). The oil can control dust, act as a lubricant to aid in the mixing of the ration, and reduce bloat (1).

On June 15, 2000, 3 Holstein dairy cattle were examined at the Large Animal Clinic at the Western College of Veterinary Medicine. All cows were presented with a similar history of anorexia, failure to drink, profuse diarrhea, and a cessation of milk production. The animals were also recumbent for long periods of time. The farmer reported that, in the cattle barn, he had a tank of agricultural feed grade canola oil that was used as an additive in the cows' daily ration. During the night of June 12, the 80 cows in the barn had apparently released the valve on the tank, spilling the entire contents (estimated to be 800 L) onto the barn floor. The farmer did not know how many cows had drunk the oil, since the event occurred overnight. The volume ingested was also unknown, since the tank was empty and floor was covered in oil. Nine cows were showing clinical signs. The 3 animals brought to the clinic were thought to be the worst affected.

A 2-year-old cow (cow 1) in mid-lactation was quiet and depressed. Based on the degree of sunken eyes and skin tent, it was estimated that dehydration was 10% of body weight. The feces were liquid, foul smelling, grey-green, and greasy in consistency. The heart rate was 80 beats/min, respirations were normal. The primary ruminal contractions were audible at the rate of 1 per 45 s. During the examination, the cow urinated normally. A sample was analyzed and appeared normal on gross examination. It was tested with a urine dip stick (Chemstrip 9; Boehringer Mannheim, Burlington, Ontario), and the results were unremarkable. A venous blood sample was collected and submitted for blood gas and electrolyte analysis. There was moderate metabolic acidosis (pH 7.24; base excess, -11.2 mEq/L), with normal electrolytes.

A 2-year-old cow (cow 2) in mid-lactation had clinical findings that were similar to those described for cow 1.

A 3-year-old cow (cow 3) in mid-lactation was the most severely affected. Depression was marked, the feces were foul smelling, the extremities felt cold, and she was 10% to 12% dehydrated. A venous blood gas analysis revealed a pH of 7.14 and a base excess of -13.4 mEq/L. The ruminal contents were green and foul smelling with a pH of 7.0 (Hydrion Paper; Essential Laboratory, Brooklyn, New York, USA). On microscopic examination of a sample of ruminal juice, no visible protozoan activity and multiple fat globules were observed. When the sample was left to stand in a tube for 15 min, the fat separated out to form a layer on the top.

Serum biochemical panels (consisting of electrolytes, liver enzymes, urea, and creatinine) done on cows 2 and 3 were within normal ranges.

A catheter was placed in the external jugular vein of each cow, and each cow received 36 L of lactated Ringer's solution over 4 h. Both cows also received 20 L of an oral electrolyte supplement (30 g KCl, 60 g

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NaCl, 30 g $NaHCO_3$) by stomach tube, at the completion of the IV fluid therapy. The cows were then placed in clean stalls that were bedded with straw and provided free access to hay and water. Cows 1 and 2 improved remarkably within 12 h of admission. They began to eat hay and drank a small amount of water. Milk production remained negligible. The consistency of the feces improved during the same time period.

All cows were also transfaunated with 8 L of ruminal juice on their 2nd day in the clinic. The ruminal juice was prepared from a resident donor cow with a permanent ruminal fistula. Over the next 4 d, the clinical condition of cows 1 and 2 remained similar. They ate a small amount of hay and drank small volumes of water. When clinical signs indicated, additional 20-L volumes of fluids and electrolytes were given by stomach tube. The cows failed to return to milk production. The fecal consistency was highly variable, fluctuating from normal to liquid.

Cow 3 remained depressed, despite similar fluid therapy, and showed no interest in feed. No improvement was noted in the consistency of the feces. A blood gas analysis on the 2nd day of therapy revealed no change in the metabolic acidosis, but a moderate hypokalemia (2.81 mEq/L) was present. Intravenous fluid therapy was repeated with a solution prepared from 8 L of normal saline with 8 L of isotonic (1.3%) $NaHCO_3$. The solution was also supplemented with 15 mmol KCl/L. This treatment was repeated on the following 2 d, as blood gas analysis continued to show the presence of a moderate metabolic acidosis with concurrent hypokalemia.

Two additional cows from the same farm were presented to the clinic on June 19 (day 6 after exposure). Clinical examination revealed depression, loss of appetite, cessation of milk production, and severe diarrhea. A close physical examination of both cows revealed only mild dehydration in 1 animal, which was treated with oral fluids. Both cows were admitted for observation.

On June 20 (day 7 after exposure), all animals were normally hydrated and had eaten small amounts of hay during the previous day. The diarrhea persisted in 3 of the 5 animals. Samples of ruminal juice collected from each cow had the appearance of normal ruminal contents and smelled normal. No protozoal activity was present on microscopic examination of the ruminal juice and no fat globules were seen. All animals were returned to the farm, as it was thought that further hospitalization was of no benefit and that they would recover.

During the evening of the same day, 2 affected animals that had not been seen at the clinic died unexpectedly. The following morning, the most severely affected of the cases that had been hospitalized (cow 3) also died. All 3 animals were submitted for a necropsy. The carcasses from the 2 animals that had died on the previous evening had undergone moderate autolysis. In one, no apparent lesions to account for the cause of death could be identified; in the other, gross lesions consistent with acute renal tubular necrosis were present in the kidney. No other lesions were found. The necropsy on cow 3 took place within 3 h of death. The most remarkable finding was a number of irregular coalescing pale foci within the myocardium of the wall of the left ventricle and the interventricular septum. Microscopic examination of sections of myocardium that were stained with hematoxylin and eosin (H&E) revealed scattered focal myocardial cells that were hypereosinophilic with dark, shrunken nuclei, but the findings were minimal and unremarkable. However, myocardial injury has to have been present for at least 16 h before it is visible with routine H&E stain. Therefore, further sections were stained with hematoxylin, basic fuschin, and picric acid (2), which can be used to demonstrate acute myocardial injury. Microscopic examination of these sections revealed a similar number of scattered focal myocytes stained in a manner consistent with peracute myocardial injury.

The ingestion of large quantities of polyunsaturated fat increases the body's requirements for vitamin E (3). Therefore, it was postulated that the ingestion of a large amount of canola oil might have been enough to cause acute nutritional muscular dystrophy (white muscle disease (WMD)) in animals that might have had a marginal deficiency in either vitamin E or selenium. Based upon the gross necropsy findings and before the results of the microscopic examination were available, the farmer was advised to treat the remaining affected animals with a parenteral sodium selenite preparation (Dystosel; rogar/STB, London, Ontario). The following day, blood samples were taken from all affected cattle to assess the levels of creatine kinase (CK) and aspartate amino transferase (AST) and determine if there was any evidence of muscle damage. The muscle enzymes were mildly elevated in a number of the cattle, but the levels were not high enough to be consistent with a diagnosis of WMD (range: CK 192 to 1568 U/L, normal 0 to 350 U/L; AST 87 to 127 U/L, normal 46 to 118 U/L). The half-life of the 2 enzymes in plasma is 4 h and 18 h, respectively (4). With the ingestion of the oil occurring a week before sampling, muscle damage would be expected to be ongoing, with continued elevation in serum muscle enzymes. Liver samples from all 3 dead cows were submitted for vitamin E and selenium analysis; in all cases, the levels of vitamin E and selenium were found to be within normal limits (vitamin E 3.8 to 4.82 ppm, normal 4.2 to 9.0 ppm; selenium 0.34 to 0.9 ppm, normal 0.25 to 0.75 ppm), effectively ruling out nutritional muscular dystrophy as a cause of death. A 4th cow (cow 1) died 2 d after the other deaths. It was not submitted for necropsy.

The ingestion of excessive quantities of any form of fat by ruminants will suppress ruminal function and reduce feed intake by coating the feed particles in the rumen and preventing bacterial digestion (5). An extensive search of the current literature reveals no information on other effects of the ingestion of large quantities of canola oil. Rapeseed oil from rapeseed, the precursor of canola, is known to be toxic by ingestion. This is predominately due to the large amount of erucic acid contained in rapeseed oil (approximately 50% by weight). Erucic acid is known to be directly cardiotoxic (3). However, erucic acid has been completely removed from canola oil by selective breeding programs. Canola oil contains predominately linoleic, oleic, and alpha-linolenic acids, all of which are considered essential fatty acids and a safe component of the diet.

Based on the findings from the macroscopic and microscopic examination of the ruminal contents, there appeared to be some degree of disruption of ruminal function. The continued presence of ruminal motility, except in 1 cow, was unexpected; however, ruminal motility is suppressed by pain, ruminal acidosis, ruminal distension, and abomasal distension (6). None of these were features of the described cases.

Serum biochemical analysis revealed 2 features. First, there was no grossly visible lipemia, indicating that excessive quantities of fat had not been absorbed from the intestines. However, it has been reported that unsaturated fatty acids should be 90% absorbed by the ruminant intestines (5). Second, there was no evidence of systemic effects, such as hepatic injury. This finding was confirmed at necropsy, when no evidence of fatty change in the liver was observed.

The appearance of the feces of the affected cows suggested that they were unable to adequately digest the canola oil and that it simply passed through the forestomachs, abomasum, and the small intestines. The severity of the diarrhea and the systemic acidosis suggested that the canola oil was acted upon by bacteria within the large intestines and broken down into short-chained fatty acids. The presence of such fatty acids may have caused an osmotic diarrhea, similar to that associated with infection with coronavirus in neonatal calves, with which foul-smelling feces, dehydration, and systemic acidosis are also associated (6).

The cause of the sudden death in 4 of the animals is not known. The first 2 animals that died were among those animals that were least affected after the ingestion of the canola oil. The finding of acute renal tubular necrosis is consistent with hypoxic injury due to severe dehydration and reduced renal perfusion (7), which would fit with the clinical picture seen in the other cows. However, it would not account for the sudden deaths. It is possible that the sudden death was due to cardiac arrest following a fatal dysrhythmia caused by serum electrolyte imbalances, particularly potassium. However, 2 of the animals had received intensive electrolyte therapy at the clinic and were discharged with normal electrolyte levels. Another theory would be that the ingestion of very large volumes of fatty acids in the canola oil resulted in a direct cardiotoxic effect.

Of the 5 remaining cattle with clinical signs, 4 dried off completely and only 1 started lactating again. However, 2 mo after the exposure, all have retained their pregnancies.

If veterinarians should encounter a similar situation, our only recommendation is that the animals be treated with fluid therapy to correct the dehydration and metabolic acidosis, in addition to receiving general supportive care. There may be some benefit in attempting to remove the oil from the rumen, or in using a binding agent, such as activated charcoal, to prevent its absorption; however, the value of such a treatment is unknown. Veterinarians should be aware that the syndrome is associated with a guarded prognosis and a high mortality rate. In the quantities recommended, canola oil remains a safe component of the dairy cow ration. CVJ

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